with the consequent consideration of cellular organelles as highly dynamic structures. Enzymes discussed here are the markers of cellular organelles. Ultra-structural study of cellular membranes is needed to confirm this hypothesis. However, results are expected to be helpful in manifesting the biochemical behaviour of carbonmonoxide.

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EFFECT OF *IN VIVO* MUSCULAR STIMULATIONS : V. SOME ASPECTS OF CARBOHYDRATE METABOLISM OF AMPHIBIAN BRAIN DURING SHORT TERM AND PROLONGED MUSCULAR STIMULATIONS

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ABSTRACT

The total carbohydrate level of brain tissue decreased in response to one day *in vivo* muscular stimulations. However the carbohydrate content returned to the normal level during prolonged stimulations. It appears that the tissue is involved in the carbohydrate sparing process, through inhibition of citric acid cycle enzymes and active uptake of lactic acid and amino acids towards the formation of carbohydrates. The tissue adaptability in carbohydrate metabolism during prolonged stimulations has been discussed.

INTRODUCTION

ELECTRICAL stimulation of muscle tissue or heavy exercise is known to alter the metabolism of many tissues in the animal[1-10]. However the information on brain metabolism is scanty. Hence an attempt has been made to understand some aspects of carbohydrate metabolism of brain in response to short term and prolonged muscular stimulations in intact animal.

MATERIALS AND METHODS

Frogs belonging to the species *Rana hexadactyla* (Lesson) were employed for the present study. The right gastrocnemius muscles of the animals were stimulated with electronic stimulaor (INCO/CSIO Research Stimulator-Ambala) as described earlier[9,10] for one day for one batch of experimental animals and for ten successive days for the other.

The brain tissue was isolated from control and experimebtal animals after pithing them, and placed in amphibian Ringer for recovering from shock effects and then employed for the studies.

The activity levels of succinate, malate and lactate dehydrogenases (SDH, MDH and LDH) and of glutamate dehydrogenase (GDH) were estimated by

the methods of Nachlas *et al.*[11] and Lee and Lardy[12] respectively as modified by Reddanna and Govindappa[10]. The levels of total carbohydrates (Carroll *et al.*[13]) free amino acids (Moore and Stein[14]) and lactic acid (Barker and Summerson[15] modified by Huckabee[16]) were estimated.

Results and Discussion

The results in Table I represent the extent of changes in brain carbohydrate metabolism in response to muscular stimulations of an intact animal. The brain tissue seems to exhibit an entirely different pattern of response in carbohydrate metabolism in

TABLE I

Levels of total carbohydrates, lactic acid and free amino acids (mg/gm wt) and succinate, malate, lactate and glutamate dehydrogenase activities (μm formazan/gm/hr) in the brain of control and experimental animals

COMPONENT	CONTROL	EXPERIMENTAL			
		1 day		10 days	% difference between 1st and 10 days
Total Carbohydrates	0·249 ± 0·099		0·16 ± 0·02	0·250 ± 0·08	+56·25
		—33·71 P < 0·05		+ 0·4 NS	P < 0·01
Lactic acid	1·186 ± 0·02		1·262 ± 0·05	1·148 ± 0·15	− 9·033
		+ 6·4 P < 0·001		− 3·21 NS	P < 0·05
Free amino acids	3·41 ± 0·26		3·87 ± 0·66	3·31 ± 0·24	−14·47
		+13·49 P < 0·05		− 2·93 NS	P < 0·05
SDH	17·85 ± 1·15		17·77 ± 2·13	13·11 ± 0·94	−25·93
		− 0·45 NS		−26·6 P < 0·001	P < 0·001
MDH	11·1 ± 1·24		9·12 ± 0·98	8·46 ± 1·9	− 7·24
		−17·84 P < 0·01		−23·78 P < 0·01	NS
LDH	23·92 ± 1·43		20·68 ± 1·71	23·78 ± 2·58	+14·99
		−13·55 P < 0·001		− 0·86 NS	P < 0·01
GDH	19·2 ± 3·0		14·24 ± 1·2	15·1 ± 1·4	+ 6·04
		−25·83 P < 0·001		−21·36 P < 0·001	NS

The values are mean of six observations; Mean ± SD ; + and − indicate per cent increase and decreas respectively.

'P' indicates level of significance ; 'NS' means non-significant.

comparison to other tissues of the body during muscular stimulations[9-10].

The total carbohydrate level drastically decreased after 1 day of muscular stimulation, suggesting the rapid degradation of brain carbohydrates in response to the applied stress. The lactic acid level increased as a consequence of carbohydrate degradations. However, the per cent increase of lactic acid was not in consonance with the per cent degradation of carbohydrates suggesting either its active diversion into citric acid cycle or its leakage into blood. NAD-dependent LDH activity showed considerable inhibition along with citric acid enzymes like SDH and MDH indicating lack of utilization of lactic acid through TCA cycle. The increased blood lactic acid level during this period (333% increase) suggests that brain may also play a contributory role in increasing blood lactic acid content. Since TCA cycle enzymes have been inhibited while lactic acid production and carbohydrate degradation occurred, it can be assumed that the brain tissue is favouring anaerobic carbohydrate degradation. Free amino acid level of the tissue increased suggesting the possibility of proteolysis or decreased amino acid oxidations. While the brain showed non-significant decrease in proteins (unpublished data) GDH activity showed considerable inhibition. Hence the increased amino acid level could largely be due to the decreased amino acid oxidations.

Carbohydrate sparing effect was shown by the brain tissue on prolonged muscular stimulations. The carbohydrate content returned to the normal level indicating the possibility of readjustments in the pattern of carbohydrate degradations. This observation was in consonance with the earlier reports where glycogen sparing effect of the muscle tissue during prolonged exercise has been suggested[17-19]. NAD-dependent LDH activity attained the normal level with a subsequent drop in lactic acid content suggesting the possibility of the decreased rate of carbohydrate degradation and/or increased rate of lactic acid utilization into the carbohydrate formation in comparison to one day stimulation. Both SDH and MDH activities have been inhibited with a rise in GDH activity in comparison to one day stimulation with a decrease in free amino acid level, suggesting the lower rate of TCA cycle, with increased utilization of amino acids.

In general it can be stated that the brain tissue seems to exhibit the 'carbohydrate sparing effect' during prolonged muscular stimulations.

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